PENDING CLAIMS

1. (Previously Amended) A method of inhibiting proliferation of a tumor cell comprising the step of inhibiting FoxM1B activity in the tumor cell by contacting the cell with a p19ARF protein fragment, wherein the p19ARF protein fragment has the amino acid sequence as set forth in SEQ ID NO:10.

2. (Original) The method of claim 1, wherein FoxM1B activity is inhibited by causing FoxM1B protein to localize in the cytoplasm of a tumor cell.

3. (Original) The method of claim 1, wherein FoxM1B activity is inhibited by preventing FoxM1B nuclear localization.

4. (Withdrawn) The method of claim 1, wherein FoxM1B activity is inhibited by contacting the tumor cell with a tyrphostin.

5. (Withdrawn) The method of claim 1, wherein the tyrphostin is a Jak2 kinase inhibitor.

6. (Withdrawn) The method of claim 5, wherein the Jak2 kinase inhibitor is AG490.

7. (Withdrawn) The method of claim 1, wherein the tumor cell is a benign tumor cell.

8. (Original) The method of claim 1, wherein the tumor cell is a malignant tumor cell.

9. (Original) The method of claim 1, wherein the tumor cell is of epithelial cell origin.

10. (Original) The method of claim 9, wherein the epithelial cell of origin is a liver, lung, skin, intestine, colon, spleen, prostate, breast, ovary, brain, or thymus epithelial cell .

11. (Previously Canceled)

12. (Withdrawn) The method of claim 1, wherein FoxM1B activity is inhibited by a Cdk1 inhibitor.

13. (Withdrawn) The method of claim 1, wherein FoxM1B activity is inhibited by interfering with the ability of FoxM1B to interact with p300/CBP.

14. (Withdrawn) The method of claim 1, wherein FoxM1B activity is inhibited by disrupting protein activity or interactions among proteins comprising the PI3K/PDK1 pathway.

15. (Withdrawn) The method of claim 1, wherein FoxM1B activity is inhibited by disrupting protein activity or interactions among proteins comprising the Ras/MAPK pathway.

16. (Withdrawn) The method of claim 1, wherein FoxM1B activity is inhibited by contacting a tumor cell with an antisense oligonucleotide, wherein the antisense oligonucleotide is complementary to nucleic acid sequences of RNA or double-stranded DNA that encodes FoxM1B and which inhibits FoxM1B gene expression.

17. (Withdrawn) A pharmaceutical composition comprising:

a. a peptide having an amino acid sequence as set forth in SEQ ID NO: 10, SEQ ID NO: 11, or SEQ ID NO: 12; and

b. a pharmaceutically acceptable carrier.

18. (Withdrawn) A method of inhibiting tumor growth in an animal comprising administering to the animal, having at least one tumor cell present in its body, a therapeutically effective amount of the pharmaceutical composition of claim 17.

19. (Withdrawn) A method of inhibiting tumor growth in an animal comprising administering to the animal, having at least one tumor cell present in its body, a therapeutically effective amount of a compound that inhibits FoxM1B activity.

20. (Withdrawn) The method of claim 19, wherein FoxM1B activity is inhibited by preventing FoxM1B nuclear localization.

21. (Withdrawn) The method of claim 19, wherein the compound is a tyrphostin.

22. (Withdrawn) The method of claim 21, wherein the tyrphostin is a Jak2 kinase inhibitor.

23. (Withdrawn) The method of claim 22, wherein the Jak2 kinase inhibitor is AG490.

24. (Withdrawn) The method of claim 19, wherein at least one tumor cell is a benign tumor cell.

25. (Withdrawn) The method of claim 19, wherein at least one tumor cell is a malignant tumor cell.

26. (Withdrawn) The method of claim 19, wherein at least one tumor cell is of epithelial cell origin.

27. (Withdrawn) The method of claim 26, wherein the epithelial cell of origin is a liver, lung, skin, intestine, colon, spleen, prostate, breast, ovary, brain or thymus epithelial cell.

28. (Withdrawn) The method of claim 19, wherein FoxM1B activity is inhibited by a Cdk1 inhibitor.

29. (Withdrawn) The method of claim 19, wherein FoxM1B activity is inhibited by interfering with the ability of FoxM1B to interact with p300/CBP.

30. (Withdrawn) The method of claim 19, wherein FoxM1B activity is inhibited by disrupting protein activity or interactions among proteins comprising the PI3K/PDK1 pathway.

31. (Withdrawn) The method of claim 19, wherein FoxM1B activity is inhibited by disrupting protein activity or interactions among proteins comprising the Ras/MAPK pathway.

32. (Withdrawn) A method of inhibiting tumor growth in an animal comprising administering to the animal, having at least one tumor cell present in its body, a therapeutically effective amount of an antisense oligonucleotide, for a therapeutically effective period of time, wherein the antisense oligonucleotide is complementary to nucleic acid sequences of RNA or double-stranded DNA that encodes FoxM1B and which inhibits FoxM1B gene expression.

33. (Withdrawn) The method of claim 32, wherein the antisense oligonucleotide is administered to the animal in a vector.

34. (Withdrawn) The method of claim 33, wherein the vector is a viral vector.

35. (Withdrawn) The method of claim 34, wherein the viral vector is an adenovirus vector, an adeno-associated virus vector, a retrovirus vector, herpes simplex virus vector, or vaccinia virus vector.

36. (Withdrawn) The method of claim 33, wherein the vector is delivered to the target cell within a liposome.

37. (Withdrawn) A method of identifying compounds that inhibit nuclear localization of FoxM1B protein, comprising the steps of:

a. contacting a cell with a candidate compound, wherein the cell expresses a green fluorescent protein-FoxM1B (GFP-FoxM1B) fusion protein;

b. contacting the cell with growth hormone;

c. detecting localization of the GFP-FoxM1B protein in the cells; and

d. identifying a compound as a compound that inhibits FoxM1B localization if the GFP-FoxM1B protein is localized in the cytoplasm and not the nuclei of the cells.

38. (Withdrawn) A method of identifying compounds that inhibit nuclear localization of FoxM1B protein, comprising the steps of:

a. contacting a transgenic mouse with a candidate compound, wherein the cells of the transgenic mouse express a green fluorescent protein-FoxM1B (GFP-FoxM1B) fusion protein;

b. administering growth hormone to the mouse;

c. detecting localization of the GFP-FoxM1B protein in a cell removed from the mouse; and

d. identifying a compound as a compound that inhibits FoxM1B nuclear localization if the GFP-FoxM1B protein is localized in the cytoplasm but not the nucleus of the cell that is removed from the mouse.

39. (Withdrawn) A method of identifying compounds that prevent tumor cell proliferation in an animal or human comprising the steps of:

a. contacting with a candidate compound a plurality of cells comprising a FoxM1B gene, wherein the cells express FoxM1B protein when cultured *in vitro*;

b. assaying FoxM1B localization in the cells; and

c. identifying a candidate compound when FoxM1B is localized in the cytoplasm and not in the nuclei of cells contacted with the compound but localized in the nuclei of cells not contacted with the compound.

40. (Withdrawn) A method of identifying compounds that can inhibit tumor progression in an animal or human comprising the steps of:

a. contacting with a candidate compound a plurality of cells comprising a FoxM1B gene, wherein the cells express FoxM1B protein when cultured *in vitro*;

b. assaying FoxM1B localization in the cells;

c. identifying a candidate compound when FoxM1B is localized in the cytoplasm and not in the nuclei of cells contacted with the compound but localized in the nuclei of cells not contacted with the compound; and

d. identifying a compound as a compound that can inhibit tumor progression if proliferation of tumor cells is inhibited when contacted with the compound *in vitro* or *in vivo*.

41. (Withdrawn) A method of identifying compounds that can inhibit tumor progression in an animal comprising the steps of:

a. contacting a plurality of cells that comprise a FoxM1B reporter construct with a candidate compound;

b. assaying FoxM1B activity in the cells; and

c. selecting a candidate compound when FoxM1B activity is decreased in cells contacted with the compound compared with FoxM1B activity in cells not contacted with the compound.

42. (Withdrawn) A method of inhibiting tumor cell proliferation comprising delivering to a tumor cell an antisense oligonucleotide, wherein the antisense oligonucleotide is complementary to nucleic acid sequences of RNA or double-stranded DNA that encodes FoxM1B and which inhibits FoxM1B gene expression.

43. (Withdrawn) The method of claim 42, wherein the antisense oligonucleotide is delivered to the cell in a vector.

44. (Withdrawn) The method of claim 43, wherein the vector is a viral vector.

45. (Withdrawn) The method of claim 44, wherein the viral vector is an adenovirus vector, an adeno-associated virus vector, a retrovirus vector, herpes simplex virus vector, or vaccinia virus vector.

46. (Withdrawn) The method of claim 42, wherein the antisense oligonucleotide is delivered to the target cell within a liposome.

47. (Withdrawn) A method of identifying compounds that can inhibit FoxM1B transcriptional activity or transformation in tissue culture systems comprising the steps of:

a. assaying for FoxM1B transcriptional activity using cotransfection assays in cells comprising a FoxM1B expression vector and a reporter gene; and

b. assaying for FoxM1B anchor independent growth by formation of colonies on soft agar using doxycycline inducible GFP-Foxm1b cell line.

48. (Withdrawn) The method of claim 47, wherein the FoxM1B expression vector is CMV-FoxM1B cDNA expression vector.

49. (Withdrawn) The method of claim 47, wherein the reporter gene comprises 6X FoxM1B binding site driving TATA box luciferase reporter gene.